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#24	Search **hama and muc-1**	15:57:25	4
#23	Search **hama and ca 125**	15:57:11	16
#22	Search **xenotypic antibody and T cytotoxic** Limits: **Publication Date to 1998/3/20**	14:16:11	0
#21	Search **xenotypic antibody and T helper** Limits: **Publication Date to 1998/3/20**	14:15:45	1
#20	Search **xenotypic antibody and T lymphocyte** Limits: **Publication Date to 1998/3/20**	14:15:12	0
#19	Search **xenotypic antibody and T cell** Limits: **Publication Date to 1998/3/20**	14:14:47	1
#18	Search **prostate specific antigen and idiotype** Limits: **Publication Date to 1998/3/20**	14:12:03	0
#17	Search **prostate specific antigen and anti-idiotype** Limits: **Publication Date to 1998/3/20**	14:11:56	0
#14	Search **anti-idiotypic antibody and prostate antigen** Limits: **Publication Date to 1998/3/20**	14:09:51	4
#13	Search **anti-idiotypic antibody and prostate** Limits: **Publication Date to 1998/3/20**	14:09:15	6
#12	Search **anti-idiotypic antibody and psa** Limits: **Publication Date to 1998/3/20**	14:08:50	1
#10	Search **anti-idiotypic antibody and MUC-1** Limits: **Publication Date to 1998/3/20**	14:07:05	7
#8	Search **anti-idiotypic antibody and CA 125** Limits: **Publication Date to 1998/3/20**	14:04:30	25
#6	Search **xenotypic antibody** Limits: **Publication Date to 1998/3/20**	13:59:59	6
#5	Search **xenotypic antibody and anti-idiotype** Limits: **Publication Date to 1998/3/20**	13:59:43	0
#4	Search **xenotypic antibody and psa** Limits: **Publication Date to 1998/3/20**	13:58:46	0
#3	Search **xenotypic antibody and MUC-1** Limits: **Publication Date to 1998/3/20**	13:58:37	0
#2	Search **xenotypic antibody and CA 125** Limits: **Publication Date to 1998/3/20**	13:58:28	0
#1	Search **xenotypic antibody and CA125** Limits: **Publication Date to 1998/3/20**	13:58:18	0

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Oct 30 2006 07:27:22

USPTO Patent Full-Text and Image Database

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Results of Search in US Patent Collection db for:
AN/altarex: 5 patents.
Hits 1 through 5 out of 5

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Refine Search AN/altarex

	PAT. NO.	Title
1	6,881,405	Reagents and methods for inducing an immune response to prostate specific antigen
2	6,716,966	Therapeutic binding agents against MUC-1 antigen and methods for their use
3	6,689,355	Therapeutic method and composition utilizing antigen-antibody complexation and presentation by dendritic cells
4	6,241,985	Method and composition for reconforming multi-epitopic antigens to initiate an immune response
5	6,086,873	Therapeutic composition and method of treatment

US PATENT & TRADEMARK OFFICE
PATENT APPLICATION FULL TEXT AND IMAGE DATABASE

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Results of Search in PGPUB Production Database for:
AN/altarex: 3 applications.
Hits 1 through 3 out of 3

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Refine Search AN/altarex

	PUB. APP. NO.	Title
1	20050260208	Binding agents and their use in targeting tumor cells
2	20050063976	Combination therapy for treating disease
3	20050048059	Therapeutic binding agents against MUC-1 antigen and methods for their use

USPTO Patent Full-Text and Image Database

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Results of Search in US Patent Collection db for:
((hama AND ca125) AND "idiotypic antibody"): 2 patents.
Hits 1 through 2 out of 2

Jump To

Refine Search hama AND ca125 and "idiotypic antibody"

	PAT. NO.	Title
1	6,241,985	Method and composition for reconforming multi-epitopic antigens to initiate an immune response
2	6,086,873	Therapeutic composition and method of treatment

USPTO Patent Full-Text and Image Database

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Results of Search in US Patent Collection db for:
(((hama AND "prostate specific antigen") AND antibody) AND anti-idiotypic): 4 patents.
Hits 1 through 4 out of 4

Jump To

Refine Search hama AND "prostate specific antigen" and antibody a

	PAT. NO.	Title
1	6,689,744	Notch receptor agonists and uses
2	6,331,402	Reduction of interference of immunoassays by substances derived from the framework regions of antibodies
3	6,274,118	Localization and therapy of non-prostatic endocrine cancer with agents directed against prostate specific antigen
4	6,068,830	Localization and therapy of non-prostatic endocrine cancer with agents directed against prostate specific antigen

US PATENT & TRADEMARK OFFICE
PATENT APPLICATION FULL TEXT AND IMAGE DATABASE

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Results of Search in PGPUB Production Database for:
hama AND ca125 and "idiotypic antibody": 8 applications.
Hits 1 through 8 out of 8

Jump To

Refine Search hama AND ca125 and "idiotypic antibody"

	PUB. APP. NO.	Title
1	20060159688	Method for diagnosing efficacy of xenotypic antibody therapy
2	20050169932	Uses of monoclonal antibody 8h9
3	20050031619	Therapeutic compositions that alter the immune response
4	20030103963	Uses of monoclonal antibody 8H9
5	20020102264	Uses of monoclonal antibody 8H9
6	20020048586	THERAPEUTIC COMPOSITIONS THAT ALTER THE IMMUNE RESPONSE
7	20020022235	Method for diagnosing efficacy of xenotypic antibody therapy
8	20010036457	Method and composition for reconforming multi-epitopic antigens to initiate an immune response

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Results of Search in PGPUB Production Database for:
hama AND "idiotypic antibody" and muc-1: 53 applications.
Hits 51 through 53 out of 53

Prev. 50 Hits

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Refine Search hama AND "idiotypic antibody" and muc-1

	PUB. APP. NO.	Title
51	20020022235	Method for diagnosing efficacy of xenotypic antibody therapy
52	20010036457	Method and composition for reconforming multi-epitopic antigens to initiate an immune response
53	20010021381	Anti-TNFalpha antibodies in therapy of asthma

US PATENT & TRADEMARK OFFICE

PATENT APPLICATION FULL TEXT AND IMAGE DATABASE

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Results of Search in PGPUB Production Database for:
hama AND "idiotypic antibody" and "prostate specific antigen": 10 applications.
Hits 1 through 10 out of 10

Jump To

Refine Search hama AND "idiotypic antibody" and "prostate specific

	PUB. APP. NO.	Title
1	20060159688	Method for diagnosing efficacy of xenotypic antibody therapy
2	20060147375	Antibodies and related molecules that bind to PSCA proteins
3	20050221400	Antibodies and related molecules that bind to PSCA proteins
4	20050142539	Targeted ligands
5	20050118164	Targeted ligands
6	20050069549	Targeted ligands
7	20050031619	Therapeutic compositions that alter the immune response
8	20040019915	Nucleic acid and corresponding protein entitled 213P1F11 useful in treatment and detection of cancer
9	20020048586	THERAPEUTIC COMPOSITIONS THAT ALTER THE IMMUNE RESPONSE
10	20020022235	Method for diagnosing efficacy of xenotypic antibody therapy

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               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
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L1 12 XENOTYPIC ANTIBODY

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L2 12621 IDIOTYPE OR IDIOTYPIC

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L3 2 L1 AND L2

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L4 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 2006:708190 CAPLUS
DN 145:123046
TI Method for diagnosing efficacy of **xenotypic antibody** therapy
IN Madiyalakan, Ragupathy; Noujaim, Antoine A.; Baum, Richard P.
PA Can.
SO U.S. Pat. Appl. Publ., 15 pp., Cont.-in-part of U.S. Ser. No. 871,339.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2006159688	A1	20060720	US 2004-824554	20040414
	WO 9742973	A1	19971120	WO 1996-IB461	19960515
	W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	JP 2001055341	A2	20010227	JP 2000-200702	19960515
	NZ 503032	A	20011130	NZ 1996-503032	19960515
	EP 1297846	A1	20030402	EP 2002-18963	19960515
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, AL				
	PT 910407	T	20030731	PT 1996-913660	19960515
	ES 2193240	T3	20031101	ES 1996-913660	19960515
	US 6241985	B1	20010605	US 1998-913290	19980320
	US 2002022235	A1	20020221	US 2001-779439	20010208
	US 2001036457	A1	20011101	US 2001-871339	20010531
	JP 2004002481	A2	20040108	JP 2003-315495	20030908
	AU 2006203074	A1	20060810	AU 2006-203074	20060718
PRAI	WO 1996-IB461	A	19960515		

	US 1998-913290	A1	19980320
	US 2000-181008P	P	20000208
	US 2001-779439	B1	20010208
	US 2001-871339	A2	20010531
	CA 1996-2253602	A	19960515
	EP 1996-913660	A3	19960515
	JP 1997-540681	A3	19960515
	JP 2000-200702	A3	19960515
	NZ 1996-332588	A1	19960515
	US 2000-201868P	P	20000504
	AU 2001-40982	A3	20010208

AB The authors disclose methods and compns. for intiating and/or enhancing an immune response by contacting an antibody with a sol. antigen. The resulting immune complex generates an enhanced immune response to the antigen. In one example, the authors demonstrate an enhanced T-cell response to an immune complex of sol. CA125 and the B43.13 monoclonal antibody.

L4 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 2001:598293 CAPLUS
DN 135:179715
TI Method for diagnosing efficacy of **xenotypic antibody** therapy
IN Noujaim, Antoine
PA Altarex Corp., Can.
SO PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001059452	A2	20010816	WO 2001-IB423	20010208
	WO 2001059452	A3	20020718		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	JP 2001055341	A2	20010227	JP 2000-200702	19960515
	NZ 503032	A	20011130	NZ 1996-503032	19960515
	EP 1297846	A1	20030402	EP 2002-18963	19960515
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, AL				
	PT 910407	T	20030731	PT 1996-913660	19960515
	ES 2193240	T3	20031101	ES 1996-913660	19960515
	CA 2399067	AA	20010816	CA 2001-2399067	20010208
	AU 2001040982	A5	20010820	AU 2001-40982	20010208
	EP 1254374	A2	20021106	EP 2001-912066	20010208
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2003522774	T2	20030729	JP 2001-558731	20010208
	JP 2004002481	A2	20040108	JP 2003-315495	20030908
	AU 2006203074	A1	20060810	AU 2006-203074	20060718
PRAI	US 2000-181008P	P	20000208		
	US 2000-201868P	P	20000504		
	EP 1996-913660	A3	19960515		
	JP 1997-540681	A3	19960515		
	JP 2000-200702	A3	19960515		
	NZ 1996-332588	A1	19960515		
	AU 2001-40982	A3	20010208		

WO 2001-IB423 W 20010208

AB The invention provides methods for diagnosing the efficacy of a patient to **xenotypic antibody** therapy which include (1) measuring the level of an antibody produced by a patient that specifically binds to a **xenotypic antibody** after administration of the **xenotypic antibody** to the patient; (2) measuring the level of an anti-**idiotypic** antibody produced by a patient that specifically binds to a **xenotypic antibody** after administration of the **xenotypic antibody** to the patient; (3) measuring the level of an antibody produced by a patient that specifically binds to a target antigen of a **xenotypic antibody** after administration of a **xenotypic antibody** to the patient; and (4) measuring the level of a T cell response produced by a patient to a target antigen of the **xenotypic antibody** after administration of a **xenotypic antibody** to the patient. In the methods of the invention, an increase in the level of antibody or T cell response produced by the patient after the administration of the **xenotypic antibody** relative to the level antibody or T cell response produced by the patient prior to the administration of the **xenotypic antibody** is indicative of a favorable diagnosis of efficacy.

=> s xenotype and antibody
L5 3 XENOTYPE AND ANTIBODY

=> duplicate remove l5
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHDS, ESBIOBASE'
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L6 1 DUPLICATE REMOVE L5 (2 DUPLICATES REMOVED)

=> d l6 bib abs 1

L6 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1

Full Text | Citing References

AN 2005:1294599 CAPLUS
DN 144:218935
TI Fluorescently tagged canine adenovirus via modification with protein IX-enhanced green fluorescent protein
AU Le, Long P.; Li, Jing; Ternovoi, Vladimir V.; Siegal, Gene P.; Curiel, David T.
CS Division of Human Gene Therapy, Departments of Medicine, Pathology and Surgery, University of Alabama at Birmingham, Birmingham, AL, 35294, USA
SO Journal of General Virology (2005), 86(12), 3201-3208
CODEN: JGVIAY; ISSN: 0022-1317
PB Society for General Microbiology
DT Journal
LA English
AB Canine adenovirus type 2 (CAV2) has become an attractive vector for gene therapy because of its non-pathogenicity and the lack of pre-existing neutralizing **antibodies** against this virus in the human population. Addnl., this vector has been proposed as a conditionally replicative adenovirus agent under the control of an osteocalcin promoter for evaluation in a syngeneic, immunocompetent canine model with spontaneous osteosarcoma. In this study, a CAV2 vector labeled with the fluorescent capsid fusion protein IX-enhanced green fluorescent protein (pIX-EGFP) was developed. Expression of the fluorescent fusion-protein label in infected cells with proper nuclear localization, and incorporation into virions, could be detected. The labeled virions could be visualized by fluorescence microscopy; this was applicable to the tracking of CAV2 infection, as well as localizing the distribution of the vector in tissues. Expression of pIX-EGFP could be exploited to detect the replication and spread of CAV2. These results indicate that pIX can serve as a platform for incorporation of heterologous proteins in the context of a canine adenovirus **xenotype**. It is believed that capsid-labeled CAV2 has utility for vector-development studies and for monitoring CAV2-based

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     oncolytic adenovirus replication.
RE.CNT  45    THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> s xenogeneic antibody and
MISSING TERM AFTER ANTIBODY AND
Operators must be followed by a search term, L-number, or query name.

=> s xenogeneic antibody
L7          79 XENOGENEIC ANTIBODY

=> s l7 and idiotype or anti-idiotypic
L8        5894 L7 AND IDIOTYPE OR ANTI-IDIOTYPIC

=> s l7 and anti-idiotypic
L9           8 L7 AND ANTI-IDIOTYPIC

=> duplicate remove l9
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L10           8 DUPLICATE REMOVE L9 (0 DUPLICATES REMOVED)

=> d l10 bib abs 1-8

L10  ANSWER 1 OF 8  CAPLUS  COPYRIGHT 2006 ACS on STN
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Full Text | Citing References

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AN   2005:343313  CAPLUS
DN   142:461722
TI   SDR grafting-a new approach to antibody humanization
AU   Kashmiri, Syed V. S.; De Pascalis, Roberto; Gonzales, Noreen R.; Schlom,
     Jeffrey
CS   Laboratory of Tumor Immunology and Biology, Center for Cancer Research,
     National Cancer Institute, National Institutes of Health, Bethesda, MD,
     20892, USA
SO   Methods (San Diego, CA, United States) (2005), 36(1), 25-34
     CODEN: MTHDE9; ISSN: 1046-2023
PB   Elsevier
DT   Journal; General Review
LA   English
AB   A review.  A major impediment to the clin. utility of the murine
     monoclonal antibodies is their potential to elicit human anti-murine
     antibody (HAMA) response in patients.  To circumvent this problem, murine
     antibodies have been genetically manipulated to progressively replace
     their murine content with the amino acid residues present in their human
     counterparts.  To that end, murine antibodies have been humanized by
     grafting their complementarity detg. regions (CDRs) onto the variable
     light (VL) and variable heavy (VH) frameworks of human Ig mols., while
     retaining those murine framework residues deemed essential for the
     integrity of the antigen-combining site.  However, the xenogeneic CDRs of
     the humanized antibodies may evoke anti-idiotypic (anti-Id) response
     in patients.  To minimize the anti-Id response, a procedure to humanize
     xenogeneic antibodies has been described that is based on grafting,
     onto the human frameworks, only the specificity detg. residues (SDRs), the
     CDR residues that are most crucial in the antibody-ligand interaction.
     The SDRs are identified through the help of the database of the
     three-dimensional structures of the antigen-antibody complexes of known
     structures or by mutational anal. of the antibody-combining site.  An
     alternative approach to humanization, which involves retention of more CDR
     residues, is based on grafting of the 'abbreviated' CDRs, the stretches of
     CDR residues that include all the SDRs.  A procedure to assess the
     reactivity of the humanized antibody to sera from patients who had been
     administered the murine antibody has also been described.
RE.CNT  40    THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD
```

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L10 ANSWER 2 OF 8 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN

Full Text

AN 2004-12683 BIOTECHDS

TI New antibodies 34B1, 26H7, and 5F4 that modulate T cell activity, useful for modulating an immune response, for treating graft versus host disease, sepsis or tumors, and for improving wound healing; monoclonal antibody preparation by hybridoma cell culture for disease therapy

AU BLUMBERG R S; BHAN A

PA BLUMBERG R S; BHAN A

PI US 2004047858 11 Mar 2004

AI US 2002-241369 11 Sep 2002

PRAI US 2002-241369 11 Sep 2002; US 2002-241369 11 Sep 2002

DT Patent

LA English

OS WPI: 2004-226167 [21]

AN 2004-12683 BIOTECHDS

AB DERWENT ABSTRACT:

NOVELTY - An antibody that modulates T cell activity selected from monoclonal antibody 34B1, 26H7, and 5F4 as produced by hybridoma 34B1, 26H7 and 5F4, respectively, deposited with ATCC (accession numbers not given), on September 4, 2002, or their antigen-binding fragment.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) a monoclonal antibody or its antigen-binding fragment that modulates T cell activity, and competes with the antibody defined above for binding to the N-domain of BGP (C-CAM1); (2) a hybridoma that produces a monoclonal antibody above; (3) a polynucleotide encoding at least a variable region of an immunoglobulin chain of any of the antibodies above; (4) a vector comprising the polynucleotide, optionally in combination with a polynucleotide that encodes the variable region of the other immunoglobulin chain of the antibody; (5) a host cell comprising a polynucleotide or vector; (6) a method for preparing an antibody or its functional fragment or immunoglobulin chain(s); (7) an antibody, its immunoglobulin chain or binding fragment encoded by a polynucleotide above, or obtainable by the method of (6); (8) a composition comprising any of the above antibody or antigen-binding fragment, polynucleotide, vector of cell; (9) a diagnostic composition comprising any of the above defined antibody, polynucleotide, vector or cell, and optionally reagents conventionally used in immuno- or nucleic acid based diagnostic methods; (10) a method of modulating the immune response in a subject by administering an antibody defined above; (11) a method of diagnosing and/or treatment of a disorder related to the neo-expression or malfunction of BGP(C-CAM1) by administering a ligand binding molecule comprising at least one CDR of an antibody above, or a corresponding **anti-idiotypic** anti-body; and (12) a method of targeting a therapeutic and/or diagnostic agent to a cell which expresses BGP(C-CAM1) or its fragment, by administering a ligand binding molecule comprising at least one CDR of an antibody above.

BIOTECHNOLOGY - Preferred Antibody: The monoclonal antibody or antigen-binding fragment is capable of inhibiting T cell proliferation in an allogenic mixed lymphocyte reaction (MLR) and/or suppressing the cytolytic activity of intestinal intraepithelial lymphocytes (iIELs). The antibody is a human, humanized, xenogeneic, or a chimeric human-murine antibody. The antigen-binding fragment is selected from a single chain Fv fragment, an F(ab') fragment, an F(ab) fragment, and an F(ab')2 fragment. The ligand-binding molecule is an antibody defined above or its immunoglobulin chain. Preferred Polynucleotide: The polynucleotide of (3) encodes at least a variable region of an immunoglobulin chain of the antibody defined above, where the variable region comprises at least one complementarity determining region (CDR) of the VH and/or VL of the variable region of the antibody. Preparation (claimed): The antibody is prepared by culturing the cell of (5), and isolating the antibody, its functional fragment or immunoglobulin chain(s) from the culture.

Preferred Composition: The composition is a pharmaceutical composition and further comprises a pharmaceutical carrier, and an immunosuppressive agent.

ACTIVITY - Immunomodulator; Immunosuppressive; Cytostatic; Antibacterial; Vulnerary. No biological data given.

MECHANISM OF ACTION - Vaccine; T cell Activator.

USE - The antibody is useful for modulating an immune response, or in the preparation of a pharmaceutical composition for modulating an immune response. The antibody, ligand binding molecule, or its corresponding **anti-idiotypic** antibody can be used for diagnosing and/or treating a disorder related to the neo-expression or malfunction of BGP(C-CAM1), or for targeting a therapeutic and/or diagnostic agent to a cell which expresses BGP(C-CAM1) or its fragment (all claimed). Specifically, the antibody is useful for treating graft versus host disease, sepsis or tumors; and for improving wound healing.

ADMINISTRATION - The antibody or composition comprising the antibody is administered intravenously, intramuscularly, subcutaneously, intraperitoneally, as an aerosol (claimed), topically or intradermally at a dose of 0.01 microg-10 mg/kg body weight.

EXAMPLE - The 34B1, 26H7 and 5F4 monoclonal antibodies (mAbs) were produced by immunizing a BALB/c mice with the activated human mucosal lymphocyte line 191 E. Three intraperitoneal injections and a final intravenous injection of 5x106 lymphocytes were given at 2-week intervals. Three days after intravenous immunization, splenocytes were isolated and fused with NS 1 murine myeloma cells in the presence of PEG. Hybridomas were selected with aminopterin-containing medium, and hybridoma supernatants were screened by indirect immunoperoxidase staining frozen intestinal and tonsillar tissue sections. Positive hybridomas were subcloned twice by limiting dilution, and ascites containing the antibody was produced by intraperitoneal injection of the hybridoma cells into pristane-treated BALB/c mice. Isotypes of 34B1 (IgG1), 26H7 (IgG1), and 5F4 (IgG1) were determined by ELISA using murine isotype-specific mAb. mAbs were purified affinity purification and protein-A sepharose columns by standard methods. (16 pages)

L10 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 2002:754149 CAPLUS
DN 137:261887
TI Antibodies and immune complexes for enhancement of the immune response to tumor
IN Schultes, Birgit C.; Nicodemus, Christopher F.
PA Altarex Corp., USA
SO PCT Int. Appl., 111 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002076384	A2	20021003	WO 2002-US7272	20020308
	WO 2002076384	A3	20030501		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
	CA 2441393	AA	20021003	CA 2002-2441393	20020308
	GB 2390811	A1	20040121	GB 2003-24503	20020308
	GB 2390811	B2	20060118		
	GB 2413960	A1	20051116	GB 2005-14967	20020308
	US 2005031619	A1	20050210	US 2004-472167	20040402
PRAI	US 2001-277599P	P	20010321		
	GB 2003-24503	A3	20020308		

WO 2002-US7272 W 20020308
AB The authors disclose the use of antibodies and immune complexes to enhance the immunogenicity of the host for tumor antigens.

L10 ANSWER 4 OF 8 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science

Full Text | Citing References

B.V. on STN
AN 2001059228 ESBIOBASE
TI Development of a minimally immunogenic variant of humanized anti-carcinoma monoclonal antibody CC49
AU Kashmiri S.V.S.; Iwahashi M.; Tamura M.; Padlan E.A.; Milenic D.E.; Schlom J.
CS S.V.S. Kashmiri, Laboratory Tumor Immunology/Biology, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, United States.
E-mail: sk85f@nih.gov
SO Critical Reviews in Oncology/Hematology, (2001), 38/1 (3-16), 90 reference(s)
CODEN: CCRHEC ISSN: 1040-8428
PUI S1040842800001335
DT Journal; General Review
CY Ireland
LA English
SL English
AB Monoclonal antibody (MAb) CC49 reacts with a pancarcinoma antigen, tumor associated glycoprotein (TAG)-72. To circumvent human anti-murine antibody (HAMA) responses in patients, we earlier developed a humanized CC49 (HuCC49) by grafting the complementarity-determining regions (CDRs) of MAb CC49 onto variable light (VL) and variable heavy (VH) frameworks of the human MAbs LEN and 21/28'CL, respectively. With the aim of minimizing its immunogenicity further, we have now generated a variant HuCC49 MAb by grafting the specificity-determining residues (SDRs) of MAb CC49 onto the frameworks of the human MAbs. Based on the evaluation of its binding affinity for TAG-72 and its reactivity with **anti-idiotypic** antibodies present in sera from patients who have been treated with murine CC49, this variant retains its antigen-binding activity and shows minimal reactivity with **anti-idiotypic** antibodies in patients' sera. Development of this variant, which is a potentially useful clinical reagent for diagnosis and therapy of human carcinomas, demonstrates that for humanization of a **xenogeneic antibody** grafting of the potential SDRs should be sufficient to retain its antigen-binding properties. Copyright © 2001 Elsevier Science Ireland Ltd.

L10 ANSWER 5 OF 8 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text | Citing References

STN
AN 1991:21244954 BIOTECHNO
TI Oral immunization with **xenogeneic antibodies** stimulates the production of systemic and mucosal **anti-idiotypic** antibodies
AU Collins A.M.; Roberton D.M.; Hosking C.S.; Flannery G.R.
CS Dept. of Microbiology/Immunol., Univ. of New South Wales, P.O. Box 1,Kensington, NSW 2033, Australia.
SO Immunology, (1991), 73/4 (388-393)
CODEN: IMMUAM ISSN: 0019-2805
DT Journal; Article
CY United Kingdom
LA English
SL English
AB The humoral and mucosal immune responses to oral immunization with **xenogeneic antibodies** were studied using an animal model in which female rabbits were fed daily doses of the MOPC-315 murine IgA antibody, and were mated during the course of the feeding programme. Serum and and colostrum samples were assayed for the presence of **anti-idiotypic** antibodies by ELISA assay, before and after depletion of anti-IgA

antibodies, by affinity chromatography using another murine IgA idiotype. It was shown that all animals responded to exposure to the MOPC-315 idiotype with the production of serum anti-murine immunoglobulin antibodies and that four of six animals produced serum **anti-idiotypic** antibodies. That the immune response included antibodies directed against the antigen-binding site was confirmed by competition ELISA assay. Mucosal IgG and IgA anti-immunoglobulin antibodies were present in milk from all antibody-fed rabbits tested, and IgA **anti-idiotypic** antibodies were detectable in the colostrum of one rabbit. The results provide some support for the hypothesis that human exposure to **xenogeneic antibodies**, most commonly bovine milk immunoglobulins, may provoke the production of **anti-idiotypic** antibodies, and that such exposure may lead to disturbances of immune regulation.

L10 ANSWER 6 OF 8 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN

Full Text | Citing References

AN 1988:19044855 BIOTECHNO
TI Problems and prospects in the use of lymphoma idiotypes as therapeutic targets
AU Stevenson G.T.; Glennie M.J.; Hamblin T.J.; Lane A.C.; Stevenson F.K.
CS Lymphoma Research Unit, Tenovus Laboratory, General Hospital, Southampton SO9 4XY, United Kingdom.
SO International Journal of Cancer, (1988), 42/SUPPL. 3 (9-12)
CODEN: IJCNAW ISSN: 0020-7136
DT Journal; Article
CY United States
LA English
SL English
AB The infusion of **anti-idiotypic** antibody in patients with lymphoma has proved a relatively innocuous procedure, but in general has yielded only partial, short-lived remissions of disease. A major problem is that **xenogeneic antibody** is simply not well-fitted to destroying mammalian cells: complement and cellular effectors (K cells and phagocytes) are not efficiently recruited, and the target cells in any case present some excellent defense mechanisms. Antigenic modulation is particularly prominent among these defenses, and we present evidence here for modulation of idiotype being much more efficient in vivo than in vitro. Two broad types of antibody derivative are under development to improve the killing of neoplastic targets. One type relies on recruiting natural effectors, and is exemplified by univalent chimeric antibody. The other relies on delivering an exogenous effector such as a drug or toxin, and is exemplified by bispecific anti-Id/anti-saporin $F(ab'\gamma)_2$ antibody. Both types of derivative have been able to suppress animal lymphoma to the extent that tumor escape occurs largely through the emergence of idiotype-negative mutants.

L10 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 1985:594621 CAPLUS
DN 103:194621
TI Monoclonal anti-idiotype antibodies against the murine B cell lymphoma 38C13: characterization and use as probes for the biology of the tumor in vivo and in vitro
AU Maloney, David G.; Kaminski, Mark S.; Burowski, Daphna; Haimovich, Joseph; Levy, Ronald
CS Med. Sch., Stanford Univ., Stanford, CA, 94305, USA
SO Hybridoma (1985), 4(3), 191-209
CODEN: HYBRDY; ISSN: 0272-457X
DT Journal
LA English
AB To establish a murine model for the monoclonal anti-idiotype (Id) immunotherapy of B cell lymphoma, a panel of rat and murine monoclonal anti-Id antibodies of several different isotypes was generated against the

surface Ig of the murine B cell tumor 38C13 (38C). **Xenogeneic antibodies** were made from fusions of rat spleen cells immunized with the 38C Id. Syngeneic monoclonal anti-idiotypes were generated from mice immunized with the Id conjugated to keyhole limpet hemocyanin. Small differences were noted in the ability of the antibodies to cross-block one another, but all appeared to be directed against the same or closely spaced idiotopes on the immunoglobulin mol. The antibodies selectively pptd. surface Ig from 38C tumor cells and not from normal mouse spleen cells. They were used to selectively stain 38C tumor cells in cell suspensions for fluorimetric anal. or immunohistochem. staining of tissue sections from mice bearing the tumor. As the malignancy progressed, the no. of tumor cells found in all tissues examd. increased. Thus, the anti-Id antibodies provided a specific probe for tumor cell detection. The antibodies had no detectable effect on cell growth in vitro; however, they did cause the rapid transient loss of the expression of cell surface Ig. This modulation was concn. and time dependent but not 100% complete. Re-expression of the Id occurred by 24 h following removal of the anti-Id antibodies. When these antibodies were used in sensitive radioisotope and enzyme linked immunoassays, the tumor cells were found to secrete small amts. of Id in vitro and in vivo. The level of Id detected in vivo correlated with tumor growth and inversely with survival.

L10 ANSWER 8 OF 8 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN

Full Text | Citing References

AN 1983:13060779 BIOTECHNO
TI Site-directed chemotherapy with a drug bound to **anti-idiotypic** antibody to a lymphoma cell-surface IgM
AU Hurwitz E.; Kashi R.; Burowsky D.; et al.
CS Dep. Chem. Immunol., Weizmann Inst. Sci., Rehovot 76100, Israel.
SO International Journal of Cancer, (1983), 31/6 (745-748)
CODEN: IJCNAW
DT Journal; Article
CY Switzerland
LA English
AB **Xenogeneic antibodies** against the cell-surface IgM of a B-cell lymphoma (38C-13) were coupled through a dextran bridge to the anti-neoplastic drug, daunomycin. The conjugate maintained both its antibody and its drug activity. The effectiveness of the conjugate was tested in vivo in mice challenged with the 38C-13 lymphoma. Drug conjugates of the idiotypic antibodies injected intraperitoneally 2 days after tumor transplantation almost completely inhibited tumor development. The controls, daunomycin-dextran-goat anti-DNP, free daunomycin, or the antibodies alone had no effect or only delayed the tumor development.

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=> s hama and ca 125
L11          38 HAMA AND CA 125

=> s l11 and (idiotype or idiotypic)
L12           9 L11 AND (IDIOTYPE OR IDIOTYPIC)

=> duplicate remove l12
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO, ESBIOBASE'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L12
L13            9 DUPLICATE REMOVE L12 (0 DUPLICATES REMOVED)

=> d l13 bib abs 1-9

L13  ANSWER 1 OF 9  CAPLUS  COPYRIGHT 2006 ACS on STN
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Full Text | Citing References

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AN   1999:811105  CAPLUS
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DN 132:49023
TI Therapeutic compositions that produce an immune response by altering the antigen
IN Madiyalakan, Ragupathy; Schultes, Birgit; Baum, Richard P.; Noujaim, Antoine A.; Leveugle, Beatrice; Kreutz, Fernando T.
PA Altarex Corp., Can.
SO PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9965517	A2	19991223	WO 1999-IB1114	19990615
	WO 9965517	A3	20000203		
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	JP 2001055341	A2	20010227	JP 2000-200702	19960515
	NZ 503032	A	20011130	NZ 1996-503032	19960515
	EP 1297846	A1	20030402	EP 2002-18963	19960515
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, AL				
	PT 910407	T	20030731	PT 1996-913660	19960515
	ES 2193240	T3	20031101	ES 1996-913660	19960515
	ZA 9810275	A	20000612	ZA 1998-10275	19981110
	CA 2333221	AA	19991223	CA 1999-2333221	19990615
	AU 9941593	A1	20000105	AU 1999-41593	19990615
	AU 762699	B2	20030703		
	EP 1085902	A2	20010328	EP 1999-925215	19990615
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002518342	T2	20020625	JP 2000-554395	19990615
	US 2002048586	A1	20020425	US 1999-376604	19990818
	JP 2004002481	A2	20040108	JP 2003-315495	20030908
PRAI	US 1998-94598	A	19980615		
	US 1998-152698	A	19980902		
	EP 1996-913660	A3	19960515		
	JP 1997-540681	A3	19960515		
	JP 2000-200702	A3	19960515		
	NZ 1996-332588	A1	19960515		
	WO 1996-IB461	A2	19960515		
	US 1997-877511	A2	19970617		
	WO 1999-IB1114	W	19990615		

AB The invention concerns methods and compns. for stimulating a host's immune response (cellular and humoral immune response), particularly for the treatment of cancer. The methods and compns. according to the invention use binding agents to generate an immune response to a predetd. sol. antigen, esp. a tumor-assocd. antigen such as CA125, CA19.9 and CA15.3. The binding agent is an antibody, a monoclonal antibody or murine monoclonal antibody that does not induce **HAMA** (human anti-murine antibodies) in patient or in the host. In accordance with the invention, the binding agent-sol. antigen complex alters the immunogenic condition of the host by generating new immunogens that are recognizable by the immune system. This leads to a humoral and/or cellular immune response.

L13 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 1998:533699 CAPLUS
DN 129:243768

TI Interferences with two-site immunoassays by human anti-mouse antibodies formed by patients treated with monoclonal antibodies: comparison of different blocking reagents
AU Reinsberg, Jochen
CS Zentrum fur Frauenheilkunde und Geburtshilfe, Universitat Bonn, Bonn, D-53127, Germany
SO Clinical Chemistry (Washington, D. C.) (1998), 44(8, Pt. 1), 1742-1744
CODEN: CLCHAU; ISSN: 0009-9147
PB American Association for Clinical Chemistry
DT Journal
LA English
AB The most widely used approach for reducing interferences with two-site immunoassays by human anti-mouse antibodies (**HAMAs**) developed by patients after exposure to murine Ig is to include high amts. of non-specific mouse IgG within the assay buffer. Recently, a polymd. form of murine IgG (MAK33) was reported to be superior to normal mouse IgG for blocking **HAMA** interference. In contrast, the authors obsd. false pos. values for the cancer antigen CA125 because of **HAMA** interference in samples from ovarian cancer patients treated with the anti-**CA 125** antibody OC125. This interference could be cor. by preincubation with mouse IgG but not with MAK33. The aim of the present study was to examine whether treatment with other monoclonal antibodies gives rise to addnl. **HAMAs** that are insensitive to MAK33 and to further characterize this specific **HAMA** response.
RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 9 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN

Full Text | Citing References

AN 1997202196 ESBIOBASE
TI ACS OV: An alternative to well established tests also measuring **CA 125**?
ACS OV: Eine Alternative zu etablierten Tests fur die Messung von **CA 125**?
AU Roth H.-J.; Zahn I.
CS H.-J. Roth, Laborgruppe Heidelberg, Abt Endokrinol Onkol Radioimmunol, Im Breitspiel 15, D-69126 Heidelberg, Germany.
E-mail: pablolim@aol.com
SO Clinical Laboratory, (1997), 43/6 (515-526), 27 reference(s)
CODEN: CLLAFP ISSN: 1433-6510
DT Journal; Article
CY Germany, Federal Republic of
LA German
SL English; German
AB The objective of the study was to evaluate the validity of the newly developed test ACS OV on the fully automated chemiluminescence analyser ACS:180 Plus. 246 patient samples with **CA 125** values covering the entire measuring range were taken out of the daily laboratory routine and analysed with the ACS OV method and 4 commercially available well established tests also measuring **CA 125** in serum and plasma. Samples were stored deep frozen at -20°C until parallel measurement was performed. Additionally, 20 selected patient samples previously measured with Enzymun Test® **CA 125** I generation and described as 'falsely elevated with old BMD assay', 6 patient samples with medium and high concentrations of human anti-mouse antibodies (**HAMA**), and 24 samples from apparently healthy laboratory technicians were included. Overall run imprecision of ACS OV with coefficients of variation not exceeding 4.1% was excellent for pool sera and commercially available QC-samples. The good assay precision was also shown by calculation of cumulative histograms for 294 patients samples. For 90% of these the coefficients of variation did not exceed 5% for determination in duplicate. A strong linear correlation was observed between ACS OV and the reference method Centocor CA 125II® with slopes of 0.828 (r = 0.966) and 0.853 (r = 0.897) for the entire measuring range of the reference method and up to 100 U/ml, respectively. Comparability with the competitor tests with coefficients of correlation between r = 0.966 and r = 0.990 was good too.

ACS OV tended to measure **CA 125** values about 15% lower than the reference method across the entire assay range. The overall lower values and an expected cut-off value of about 21 U/ml, based on a small number of healthy control subjects, reveals the need for further investigations to establish decision limits. It should be pointed out that the ACS OV test showed no interference even with very high levels of **HAMA** and with none of the samples which mere 'falsely elevated with old BMD assay'. Due to the selection of B27.1 as capture antibody (OC125 antigenic domain) one can assume as well that circulating anti-**idiotypic** anti-OC125 antibodies might not interfere in the test. In our estimation the newly developed ACS OV test, with its evaluated test characteristics and its excellent automated technical performance on the fully automated analyser ACS:180 Plus, is a reliable tool for monitoring ovarian cancer patients and provides a good alternative to well established tests also measuring **CA 125**.

L13 ANSWER 4 OF 9 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science

B.V. on STN

AN 1995084654 ESBIOBASE

TI Determination of the cancer antigen 125 with a second generation assay - Interferences by human anti-OC125 antibodies
BESTIMMUNG DES CANCER ANTIGEN 125 MIT DER ZWEITEN TEST-GENERATION - INTERFERENZEN DURCH HUMANE ANTI-OC125-ANTIKORPER

AU Reinsberg J.; Richter H.

CS Dr. J. Reinsberg, Zentrum Frauenheilkunde Geburtshilfe, Universitat Bonn, Sigmund-Freud-Strasse 25, D-53127 Bonn, Germany.

SO Klinisches Labor, (1995), 41/5 (345-350)
CODEN: KLLAEA ISSN: 0941-2131

DT Journal; Article

CY Germany, Federal Republic of

LA English

SL English; German

AB We examined the second generation **CA-125** assay Enzymun Test **CA-125** II, which involves enzyme-linked OC125 detector antibodies and biotinylated M11 capture antibodies, for interferences due to human anti-OC125 antibodies. Testing serum samples obtained from ovarian cancer patients treated with OC125 fragments, with anti-**idiotypic** anti-OC125 antibody concentrations ranging from 4.6×10^3 to 2.1×10^6 kU/l and non-specific human anti-mouse antibody concentrations ranging from 36 to 34830 µg/l we found falsely high **CA 125** values: in the presence of anti-mouse antibody concentrations between 180 µg/l and 320 µg/l a falsely high assay response equivalent to a **CA-125** concentration of 400 kU/l was measured. After elimination of these interferences by addition of murine IgG, an inhibitory effect of anti-OC125 antibodies became evident: from samples with anti-**idiotypic** anti-OC125 antibody concentrations exceeding 4.0×10^4 kU/l less than 22% of added **CA 125** was recovered. The present data suggest that non-specific human anti-mouse antibodies are responsible for falsely high results while the reduction of the recovery rate is due to an inhibition of binding of OC125 detector antibodies to **CA-125** by anti-**idiotypic** anti-OC125 antibodies. With the present test protocol the Enzymun Test **CA-125** II should therefore not be used for **CA-125** determination in patients treated with OC125 fragments.

L13 ANSWER 5 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

STN

AN 1995:25162739 BIOTECHNO

TI Antiidiotype induction therapy: Evidence for the induction of immune response through the **idiotype** network in patients with ovarian cancer after administration of anti-CA125 murine monoclonal antibody B43.13

AU Madiyalakan R.; Sykes T.R.; Dharampaul S.; Sykes C.J.; Baum R.P.; Hor G.; Noujaim A.A.

CS Biomira Research Inc., 1134 Dentistry Pharmacy Bldg., University of Alberta,Edmonton, Alta. T6G 2N8, Canada.
SO Hybridoma, (1995), 14/2 (199-203)
CODEN: HYBRDY ISSN: 0272-457X
DT Journal; Conference Article
CY United States
LA English
SL English
AB The immune status of ovarian cancer patients receiving anti-CA125 routine monoclonal antibody B43.13 was evaluated by measuring antiidiotypic antibodies (Ab_2), antiantiidiotypic antibodies (Ab_3), antiisotypic human antimouse antibodies (**HAMA**), interferon-γ, and CA125 levels in the serum. A specific assay was developed for the determination of Ab_2 antibodies using chimeric MAb B43.13. Of the 50 patients studied, 26 had elevated levels of Ab_2. Eleven of these 26 patients also had high titer of antiantiidiotypic (Ab_3) antibodies. Eight of the 22 patients analyzed had increased interferon-γ levels. A tentative correlation was found between survival of these patients' antiidiotype induction.

L13 ANSWER 6 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text | Citing References

STN
AN 1994:24046678 BIOTECHNO
TI Activating anti-**idiotypic** human anti-mouse antibodies for immunotherapy of ovarian carcinoma
AU Baum R.P.; Niesen A.; Hertel A.; Nancy A.; Hess H.; Donnerstag B.; Sykes T.R.; Sykes C.J.; Suresh M.R.; Noujaim A.A.; Hor G.
CS Department of Nuclear Medicine, University Medical Center, Theodor Stern Kai 7,60590 Frankfurt/Main, Germany.
SO Cancer, (1994), 73/SUPPL. (1121-1125)
CODEN: CANCAR ISSN: 0008-543X
DT Journal; Conference Article
CY United States
LA English
SL English
AB Human anti-mouse antibodies (**HAMA**) are observed frequently after immunoscintigraphy with monoclonal antibodies (MoAb) directed against **CA- 125**. As the authors have shown previously, **HAMA** can cause false-positive **CA- 125** values in routine **CA-125** immunoradiometric assay (IRMA) tumor-marker assays (in one case, up to 900 days after immunoscintigraphy). In 32 patients, the authors found a **HAMA** frequency of 34% (11/32: 3/7 after the first administration, 6/13 after the second, and 2/2 after the third). Ten patients developed extremely high **CA-125** levels after undergoing the CIS IRMA assay (up to 80,000 U/ml) in parallel to a significant **HAMA** increase. The use of different assays, or **HAMA** removal before in vitro testing, can solve this problem. After a new **CA-125** assay containing antibodies that recognize different epitopes on the **CA-125** antigen (Biomira TruQuant OV) was applied, only mildly increased assay results or normal levels were measured. Most of **HAMA**-positive patients demonstrated a predominantly anti-**idiotypic** response, determined with two different **HAMA** assays. Seven patients with anti-**idiotypic HAMA** responses after OC-125 immunoscintigraphy remained free of tumor or had stable disease (2-42 or more months), contrary to their poor prognoses that had been made based on the underlying stages of their tumors. All of these patients are currently doing well (Karnofsky Index > 70%) and show no significant tumor progression. In light of their extremely poor prognoses (5- year survival rates of 3-5% in recurrent International Federation of Gynecology and Obstetrics III/IV stages), without further chemotherapy, these courses are extremely unusual. Preliminary in vitro experiments lead to the postulatation that anti-**idiotypic HAMA** may trigger an antitumor effect either by suppressing the growth of **CA-125**-expressing cancer cells directly, or by activating the patient's immune response via induction of Ab3. Similar results are observed after

immunoscintigraphy with a technetium-99m-labeled anti-**CA-125** monoclonal antibody (B43.13), which the authors now also use for immunotherapy of ovarian cancer patients by repeated injections, hoping that induction of anti-**idiotypic HAMA** will be beneficial for prolonged survival of patients with ovarian carcinoma.

L13 ANSWER 7 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text | Citing References

STN
AN 1993:24198149 BIOTECHNO
TI Clinical course of ovarian cancer patients under repeated stimulation of **HAMA** using MAb OC125 and B43.13
AU Baum R.P.; Noujaim A.A.; Nanci A.; Moebus V.; Hertel A.; Niesen A.; Donnerstag B.; Sykes T.; Boniface G.; Hor G.
CS Department of Nuclearmedicine, University of Frankfurt, Theodor Stern Kai 7,W-6000 Frankfurt/Main, Germany.
SO Hybridoma, (1993), 12/5 (583-589)
CODEN: HYBRDY ISSN: 0272-457X
DT Journal; Conference Article
CY United States
LA English

L13 ANSWER 8 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text | Citing References

STN
AN 1992:22057371 BIOTECHNO
TI Human antibody response to the intravenous and intraperitoneal administration of the $F(ab')_2$ fragment of the OC125 murine monoclonal antibody
AU Maher V.E.; Drukman S.J.; Kinders R.J.; Hunter R.E.; Jennings J.; Brigham C.; Stevens S.; Griffin T.W.
CS Division of Oncology/Medicine, University of Massachusetts, Medical Center, 55 Lake Avenue North,Worcester, MA 01655, United States.
SO Journal of Immunotherapy, (1992), 11/1 (56-66)
CODEN: JOIME7 ISSN: 1053-8550
DT Journal; Article
CY United States
LA English
SL English
AB We have characterized the human immune response against murine monoclonal antibodies (**HAMA**) in 18 patients following administration of the $F(ab')_2$ fragment of the murine monoclonal antibody OC125. OC125 is directed against the CA125 antigen, present on the surface of many human ovarian cancers. An affinity matrix was used to separate serum into immunoglobulin-containing and immunoglobulin-free fractions. **HAMA** titer was determined on the immunoglobulin fraction with an OC125 sandwich enzyme-linked immunosorbent assay (ELISA). All patients developed an **HAMA** response, despite the use of $F(ab')_2$ fragments and small amounts (1-4 mg) of antibody. It may be that the intraperitoneal (i.p.) route provides a more marked **HAMA** response. Enzyme-linked sandwich immunoassays were also used to determine anti-isotype and anti-**idiotype** titers. Anti-isotype titers were analyzed with antigen irrelevant, isotype-matched murine antibodies and OC125-HRPO. Anti-**idiotypes** titers were assessed in a sandwich assay that utilized $F(ab')_2$ and F(ab') fragments of OC125. The anti-isotype response tended to be of low titer and short duration, while the anti-**idiotype** response was of high titer and remarkably persistent. **HAMA** interfered in an unpredictable manner with the correct measurement of serum levels of CA125 in an enzyme immunoassay using OC125. Corrected values of CA125 could be obtained by measurement of antigen in the immunoglobulin-free fraction of serum. The response of one patient, who developed a markedly elevated anti-**idiotype** titer after serial i.v./i.p. injections, was further characterized and found to contain an antibody consistent with an anti-anti-**idiotype** to the CA125 antigen.

L13 ANSWER 9 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text | Citing References

STN

AN 1990:20251933 BIOTECHNO

TI Interference by human anti-mouse antibodies in **CA 125** assay after immunoscintigraphy: Anti-**idiotypic** antibodies not neutralized by mouse IgG but removed by chromatography

AU Turpeinen U.; Lehtovirta P.; Alfthan H.; Stenman U.-H.

CS Helsinki University Central Hospital, Department of Obstetrics and Gynecology, Laboratory, Haartmaninkatu 2, 00290 Helsinki, Finland.

SO Clinical Chemistry, (1990), 36/7 (1333-1338)
CODEN: CLCHAU ISSN: 0009-9147

DT Journal; Article

CY United States

LA English

SL English

AB Falsely increased concentrations of the ovarian carcinoma-associated antigen, **CA 125**, were measured by a monoclonal antibody (MAV)-based double determinant immunoradiometric assay (IRMA) in patients who developed antibodies to mouse immunoglobulins (IgGs) after receiving injections of the same MAb as is used in the **CA 125** IRMA. Addition of undiluted mouse serum or purified mouse IgG to the assay mixture failed to eliminate the falsely increased **CA 125** concentrations in most of the samples, owing to the presence of anti-**idiotype** antibody. Because of their anti-**idiotypic** nature, the human anti-mouse antibodies (**HAMAS**) had only little effect on other immunometric assays, and this effect could be completeley eliminated by addition of mouse IgG. To eliminate the effect of **HAMA** on the **CA 125** assay, we studied the ability of various chromatographic methods to separate the interfering **HAMA** from **CA 125**. For measuring **HAMA** in serum and chromatographic fractions we developed a time-resolved fluoroimmunoassay. Adequate separation of **CA 125** and **HAMA** was achieved by affinity chromatography of patients' sera with solid-phase Protein A, Protein G, cation-exchange chromatography on Mono S, and gel filtration on Superose 6. These results demonstrate that the interference can effectively be removed by rather simple chromatographic procedures.

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=> s hama and muc-1
L14          2 HAMA AND MUC-1

=> duplicate remove l14
PROCESSING COMPLETED FOR L14
L15           2 DUPLICATE REMOVE L14 (0 DUPLICATES REMOVED)

=> s hama and muc-1
L14          2 HAMA AND MUC-1

=> d l15 bib abs 1-2
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L15 ANSWER 1 OF 2 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN

Full Text

AN 2005-09748 BIOTECHDS

TI New therapeutic composition comprising a non-radiolabeled binding agent or a binding agent other than HMFG1 that specifically binds to an epitope of soluble and tumor-bound tumor-associated **MUC-1**, useful in treating tumor in a mammal;
for use in tumor gene therapy

AU MADIYALAKAN R; QI W; SCHULTES B C

PA ALTAREX CORP

PI US 2005048059 3 Mar 2005

AI US 2004-754089 7 Jan 2004

PRAI US 2004-754089 7 Jan 2004; US 1999-149492 18 Aug 1999

DT Patent
LA English
OS WPI: 2005-181416 [19]
AN 2005-09748 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - A new therapeutic composition comprises a non-radiolabeled binding agent or a binding agent other than HMFG1 that specifically binds to an epitope of soluble and tumor-bound tumor-associated **MUC-1**.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) treating a mammal bearing a tumor; (2) a binding agent that binds immunological determinants from amino acid residues of a peptide having the amino acid sequence Asp-Thr-Arg-Pro-Ala-Pro or that binds the same epitope as Alt-1; (3) Alt-1; and (4) treating a mammal bearing a tumor, where the animal has a baseline level of anti-**MUC-1** antibody.

BIOTECHNOLOGY - Preferred Composition: The therapeutic composition comprises a non-radiolabeled binding agent or a binding agent other than HMFG1 that specifically binds to an epitope of soluble and tumor-bound tumor-associated **MUC-1**. The binding agent is not a monoclonal antibody comprising HMPV, VU-3-C6, MF06, VU-11-D1, MF30, BCP8, DF3, BC2, B27.29, VU-3-D1, 7540MR, MF11, Bc4E549, VU-11-E2, M38, E29, GP1.4,214D4, BC4W154, HMFG-2, C595, Mc5 or A76-A/C7. The binding agent induces an anti-idiotype response and a cellular immune response in the mammal. The binding agent binds immunological determinants from amino acid residues of a peptide having the amino acid sequence Asp-Thr-Arg-Pro-Ala-Pro or that binds the same epitope as Alt-1. The binding agent is photoactivated. The binding agent is coupled to a photodynamic agent. The photodynamic agents include hypocrellins or hypocrellin derivatives. The epitope comprises an immunological determinant that includes carbohydrate. Preferred Method: Treating a mammal bearing a tumor comprises administering the binding agent or therapeutic composition. The binding agent is administered at a dosage that is the maximum amount of binding agent that does not induce antibody-mediated toxicity or that does not produce ADCC or CDC. The binding agent is administered at a dosage that elicits a HAXA response greater than 200 U/ml or that reduces the level of tumor antigen CA15.3. The HAXA response is a **HAMA** response. The method further comprises irradiating the mammal with a visible light source. The binding agent is administered in the absence or presence of an adjuvant. The binding agent binds both circulating and tumor-bound **MUC-1**. Treating a mammal bearing a tumor, where the animal has a baseline level of anti-**MUC-1** antibody comprises subcutaneously administering the therapeutic composition comprising a binding agent that specifically binds to an epitope of tumor-associated **MUC-1** that causes an increase of at least 3-fold in anti-**MUC-1** antibody compared to the baseline level.

ACTIVITY - Cytostatic. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The therapeutic composition is useful in treating a mammal bearing a tumor (claimed).

ADMINISTRATION - Dosage comprises less than about 8, 3, preferably 0.5-2, 0.5-1.5 or 1 mg/30 kg body weight. The composition is administered via intravenous or subcutaneous route. (All claimed.)

EXAMPLE - No relevant examples given. (20 pages)

L15 ANSWER 2 OF 2 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN

Full Text

AN 2004-14318 BIOTECHDS
TI New binding agent, Alt-1, that binds immunological determinants of **MUC-1**, useful for therapeutically treating a mammal bearing a tumor e.g. breast, colon, esophageal, prostate or pancreatic carcinoma, or multiple myeloma;
antibody production via cell culture against **MUC-1** for use in disease therapy
AU MADIYALAKAN R
PA ALTAREX CORP
PI US 6716966 6 Apr 2004

AI US 2000-641833 18 Aug 2000
PRAI US 2000-641833 18 Aug 2000; US 1999-149492 18 Aug 1999
DT Patent
LA English
OS WPI: 2004-303095 [28]
AN 2004-14318 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - A binding agent that binds immunological determinants from amino acid residues of an epitope including carbohydrate and a peptide sequence (I) consisting of amino acid residues 3-8 of an 8-amino acid sequence (SEQ ID NO: 2) peptide, is new.

DETAILED DESCRIPTION - A binding agent that binds immunological determinants from amino acid residues of an epitope including carbohydrate and a peptide sequence (I) consisting of amino acid residues 3-8 of an 8-amino acid sequence (SEQ ID NO: 2) peptide. (I) comprises the amino acid sequence: Asp-Thr-Arg-Pro-Ala-Pro (I) INDEPENDENT CLAIMS are also included for: (1) the antibody Alt-1 produced by the hybridoma having ATCC number PTA-975; and (2) a therapeutic composition comprising a binding agent selected from Alt-1 and the binding agent defined above.

BIOTECHNOLOGY - Preferred Binding Agent: The binding agent is photoactivated. Preferred Method: In treating a mammal bearing a tumor, the binding agent is administered at a dosage that is the maximum amount of binding agent that does not induce antibody-mediated toxicity or that does not produce ADCC or CDC. The binding agent is administered at a dosage that elicits a HAXA response of more than 200 U/ml or more than 2000 U/ml, where the HAXA response is a **HAMA** response. The binding agent may also be administered at a dosage that reduces the level of tumor antigen CA15.3. The binding agent is administered in the absence or in the presence of an adjuvant. The method further comprises irradiating the mammal with a visible light source. The binding agent binds both circulating and tumor-bound **MUC-1**.

ACTIVITY - Cytostatic. MT-CB6F1 mouse tumor model administered with MAb-Alt-1 resulted in reduction of tumor burden and greater survival advantage compared with mice treated with PBS. A minimum of 3 intravenous injections prior to inoculation of the tumor cells, and 4 subsequence injections were necessary to demonstrate this effect.

MECHANISM OF ACTION - **MUC-1** Inhibitor.

USE - The composition comprising the Alt-1 antibody or the binding agent is useful for therapeutically treating a mammal bearing a tumor (claimed) e.g. breast carcinoma, colon carcinoma, esophageal squamous cell carcinoma, pancreatic carcinoma, prostate carcinoma or multiple myeloma.

ADMINISTRATION - The composition is administered at a dose of less than about 8 mg/30 kg body weight, and the binding agent is administered at a dose of less than 3 mg/30 kg body weight, such as 0.5-2 mg/30 kg body weight, preferably 1 mg/30 kg body weight. The composition or the binding agent is administered intravenously or subcutaneously (claimed).

EXAMPLE - The murine cell or hybridoma that secretes the binding agent was generated by immunizing mice with MUC1, and harvesting and immortalizing the antibody-secreting splenocytes by fusing them with the myeloma cell line SP2/0-Ag14. The desired clone was selected by assaying the secreted antibodies for the ability to bind MUC1, and selected clone was then expanded in a medium. Isotype switching of the clone from an IgM to an IgG was performed, and continued 0.2 microm filtration and dilution using formulation buffer was done. Specific binding of the binding agent to MUC1 expressing breast cancer cells and MUC1 transformed murine cells was demonstrated by FACS, fluorescence microscopy, and other binding assays. The binding agent was then purified from the growth media by standard techniques.(18 pages)

```
=> s hama and psa or (prostate specific antigen)
L16      13610 HAMA AND PSA OR (PROSTATE SPECIFIC ANTIGEN)

=> s hama and psa
L17          0 HAMA AND PSA
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=> s hama and (prostate specific antigen)
L18           5 HAMA AND (PROSTATE SPECIFIC ANTIGEN)

=> duplicate remove l18
PROCESSING COMPLETED FOR L18
L19           5 DUPLICATE REMOVE L18 (0 DUPLICATES REMOVED)

=> d l19 bib abd 1-5
'ABD' IS NOT A VALID FORMAT FOR FILE 'CAPLUS'

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CBIB -------- AN, plus Compressed Bibliographic Data
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MAX --------- ALL, plus Patent FAM, RE
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SCAN -------- CC, SX, TI, ST, IT (random display, no answer numbers;
              SCAN must be entered on the same line as the DISPLAY,
              e.g., D SCAN or DISPLAY SCAN)
STD --------- BIB, CLASS

IABS -------- ABS, indented with text labels
IALL -------- ALL, indented with text labels
IBIB -------- BIB, indented with text labels
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OIBIB ------- OBIB, indented with text labels

SBIB -------- BIB, no citations
SIBIB ------- IBIB, no citations

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              containing hit terms
HITRN ------- HIT RN and its text modification
HITSTR ------ HIT RN, its text modification, its CA index name, and
              its structure diagram
HITSEQ ------ HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
              its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
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OCC --------- Number of occurrence of hit term and field in which it occurs

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codes.  For a list of the display field codes, enter HELP DFIELDS at
an arrow prompt (=>).  Examples of formats include: TI; TI,AU; BIB,ST;
TI,IND; TI,SO.  You may specify the format fields in any order and the
information will be displayed in the same order as the format
specification.
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to view a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):d l19 bib abs 1-5
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ALL --------- BIB, AB, IND, RE
APPS -------- AI, PRAI
BIB --------- AN, plus Bibliographic Data and PI table (default)
CAN --------- List of CA abstract numbers without answer numbers
CBIB -------- AN, plus Compressed Bibliographic Data
CLASS ------- IPC, NCL, ECLA, FTERM
DALL -------- ALL, delimited (end of each field identified)
DMAX -------- MAX, delimited for post-processing
FAM --------- AN, PI and PRAI in table, plus Patent Family data
FBIB -------- AN, BIB, plus Patent FAM
IND --------- Indexing data
IPC --------- International Patent Classifications
MAX --------- ALL, plus Patent FAM, RE
PATS -------- PI, SO
SAM --------- CC, SX, TI, ST, IT
SCAN -------- CC, SX, TI, ST, IT (random display, no answer numbers;
              SCAN must be entered on the same line as the DISPLAY,
              e.g., D SCAN or DISPLAY SCAN)
STD --------- BIB, CLASS

IABS -------- ABS, indented with text labels
IALL -------- ALL, indented with text labels
IBIB -------- BIB, indented with text labels
IMAX -------- MAX, indented with text labels
ISTD -------- STD, indented with text labels

OBIB -------- AN, plus Bibliographic Data (original)
OIBIB ------- OBIB, indented with text labels

SBIB -------- BIB, no citations
SIBIB ------- IBIB, no citations

HIT --------- Fields containing hit terms
HITIND ------ IC, ICA, ICI, NCL, CC and index field (ST and IT)
              containing hit terms
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HITSEQ ------ HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
              its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
KWIC -------- Hit term plus 20 words on either side
OCC --------- Number of occurrence of hit term and field in which it occurs

To display a particular field or fields, enter the display field
codes.  For a list of the display field codes, enter HELP DFIELDS at
an arrow prompt (=>).  Examples of formats include: TI; TI,AU; BIB,ST;
TI,IND; TI,SO.  You may specify the format fields in any order and the
information will be displayed in the same order as the format
specification.

All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR,
FHITSTR, HITSEQ, FHITSEQ, KWIC, and OCC) may be used with DISPLAY ACC
to view a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):bib
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L19 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 2004:354752 CAPLUS
DN 140:355853
TI Therapeutic adjuvant activity of xenotypic antibody
IN Schultes, Birgit C.; Nicodemus, Christopher F.
PA Altarex Corp., Can.; Altarex Medical Corporation
SO PCT Int. Appl., 72 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004035003	A2	20040429	WO 2003-US33027	20031017
	WO 2004035003	A3	20050113		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2504656	AA	20040429	CA 2003-2504656	20031017
	AU 2003286463	A1	20040504	AU 2003-286463	20031017
	EP 1578445	A2	20050928	EP 2003-777662	20031017
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	US 2005271649	A1	20051208	US 2005-531849	20050715
PRAI	US 2002-419332P	P	20021017		
	WO 2003-US33027	W	20031017		

L19 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 2003:221907 CAPLUS
DN 138:234416
TI Methods and apparatus for conducting multiple measurements on a sample
IN Glezer, Eli N.; Johnson, Kent; Tsionsky, Michael; Kenten, John H.; Debad, Jeff D.; Umek, Robert M.; Eason, Paula Denney; Biebuyck, Hans; Wohlstadter, Jacob N.; Wilbur, James; Sigal, George
PA Meso Scale Technologies, LLC, USA
SO PCT Int. Appl., 147 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003023360	A2	20030320	WO 2002-US28652	20020910
	WO 2003023360	A3	20030710		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
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	CA 2459893	AA	20030320	CA 2002-2459893	20020910
	US 2003113713	A1	20030619	US 2002-238391	20020910
	US 2003207290	A1	20031106	US 2002-238960	20020910
	US 7063946	B2	20060620		
	EP 1436620	A2	20040714	EP 2002-798183	20020910
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
	JP 2005521032	T2	20050714	JP 2003-527386	20020910
	US 2005136048	A1	20050623	US 2005-54672	20050209
	US 2005136497	A1	20050623	US 2005-55472	20050209
PRAI	US 2001-318284P	P	20010910		
	US 2001-318289P	P	20010910		
	US 2001-318293P	P	20010910		
	US 2002-363498P	P	20020311		
	US 2002-409442P	P	20020909		
	US 2002-238437	A3	20020910		
	WO 2002-US28652	W	20020910		

L19 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

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AN 2001:833121 CAPLUS
DN 135:370635
TI Therapeutic application of immune complexes and their presentation by dendritic cells
IN Schultes, Birgit; Noujaim, Antoine
PA Altarex Corp., Can.
SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001085204	A2	20011115	WO 2001-IB1331	20010511
	WO 2001085204	A3	20020822		
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	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2408549	AA	20011115	CA 2001-2408549	20010511
	US 2002048583	A1	20020425	US 2001-853300	20010511
	US 2002164312	A1	20021107	US 2001-853268	20010511
	US 6689355	B2	20040210		
	EP 1294398	A2	20030326	EP 2001-949830	20010511
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2003532687	T2	20031105	JP 2001-581857	20010511
	US 2004191236	A1	20040930	US 2003-683510	20031010
PRAI	US 2000-203635P	P	20000511		
	US 2000-253671P	P	20001128		
	US 2000-253956P	P	20001128		
	US 2001-853268	A1	20010511		
	WO 2001-IB1331	W	20010511		

L19 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 2001:833120 CAPLUS
DN 135:370634
TI Therapeutic application of immune complexes and their presentation by

dendritic cells
IN Schultes, Birgit; Noujaim, Antoine; Mann, Dean
PA Altarex Corp., Can.
SO PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001085203	A2	20011115	WO 2001-IB1238	20010511
	WO 2001085203	A3	20020822		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2408547	AA	20011115	CA 2001-2408547	20010511
	US 2002048583	A1	20020425	US 2001-853300	20010511
	US 2002164312	A1	20021107	US 2001-853268	20010511
	US 6689355	B2	20040210		
	EP 1294397	A2	20030326	EP 2001-945568	20010511
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2003532686	T2	20031105	JP 2001-581856	20010511
	US 2004191236	A1	20040930	US 2003-683510	20031010
PRAI	US 2000-203635P	P	20000511		
	US 2000-253671P	P	20001128		
	US 2000-253956P	P	20001128		
	US 2001-853268	A1	20010511		
	WO 2001-IB1238	W	20010511		

L19 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 1999:811105 CAPLUS
DN 132:49023
TI Therapeutic compositions that produce an immune response by altering the antigen
IN Madiyalakan, Ragupathy; Schultes, Birgit; Baum, Richard P.; Noujaim, Antoine A.; Leveugle, Beatrice; Kreutz, Fernando T.
PA Altarex Corp., Can.
SO PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9965517	A2	19991223	WO 1999-IB1114	19990615
	WO 9965517	A3	20000203		
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	JP 2001055341	A2	20010227	JP 2000-200702	19960515
	NZ 503032	A	20011130	NZ 1996-503032	19960515
	EP 1297846	A1	20030402	EP 2002-18963	19960515

	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, AL				
	PT 910407	T	20030731	PT 1996-913660	19960515
	ES 2193240	T3	20031101	ES 1996-913660	19960515
	ZA 9810275	A	20000612	ZA 1998-10275	19981110
	CA 2333221	AA	19991223	CA 1999-2333221	19990615
	AU 9941593	A1	20000105	AU 1999-41593	19990615
	AU 762699	B2	20030703		
	EP 1085902	A2	20010328	EP 1999-925215	19990615
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002518342	T2	20020625	JP 2000-554395	19990615
	US 2002048586	A1	20020425	US 1999-376604	19990818
	JP 2004002481	A2	20040108	JP 2003-315495	20030908
PRAI	US 1998-94598	A	19980615		
	US 1998-152698	A	19980902		
	EP 1996-913660	A3	19960515		
	JP 1997-540681	A3	19960515		
	JP 2000-200702	A3	19960515		
	NZ 1996-332588	A1	19960515		
	WO 1996-IB461	A2	19960515		
	US 1997-877511	A2	19970617		
	WO 1999-IB1114	W	19990615		

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=> s alterex
L20           0 ALTEREX

=> s altarex
L21          15 ALTAREX

=> s l21 and antibody
L22          13 L21 AND ANTIBODY

=> duplicate remove l22
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO, ESBIOBASE'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L22
L23           10 DUPLICATE REMOVE L22 (3 DUPLICATES REMOVED)

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DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO, ESBIOBASE'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L21
L24           12 DUPLICATE REMOVE L21 (3 DUPLICATES REMOVED)

=> d l24 bib abs 1-12

L24  ANSWER 1 OF 12  CAPLUS  COPYRIGHT 2006 ACS on STN DUPLICATE 1
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Full Text | Citing References

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AN   2004:520066  CAPLUS
DN   141:312437
TI   Immunotherapy of ovarian cancer with antibodies: a focus on oregovomab
AU   Berek, Jonathan S.
CS   24-137 UCLA Center for the Health Sciences, David Geffen School of
     Medicine at UCLA, Los Angeles, CA, 90095-1740, USA
SO   Expert Opinion on Biological Therapy (2004), 4(7), 1159-1165
     CODEN: EOBTA2; ISSN: 1471-2598
PB   Ashley Publications Ltd.
DT   Journal; General Review
LA   English
AB   A review.  Recent advances in the mol. and cellular biol. of malignancy
     and tumor immunol. have stimulated significant progress in the application
     of immunotherapies as adjuvant treatments in cancer.  Oregovomab
     (OvaRex,AltaRex) is a murine monoclonal antibody with high affinity to
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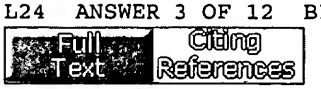
the ovarian cancer assocd. antigen CA125. Infusion of low-dose antibody results in formation of circulating immune complexes which can trigger a cellular immune response targeting CA125 and the ovarian cancer. Oregovomab has activity following initial chemotherapy and in recurrent disease settings and is in Phase III trials to establish its efficacy to prolong time to relapse in patients with advanced ovarian cancer and favorable outcomes to their front-line treatment. Addnl. studies of antigen processing and combination chemo-immunotherapy are ongoing. The treatment shows promise as a potential new addn. to the std. care of ovarian cancer.

RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 2 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

on STN
AN 2003:36553208 BIOTECHNO
TI Cancer Drug Development: New Directions and Challenges - SMi Conference: 10-11 March 2003, London, UK
AU Erlich R.
CS R. Erlich, Thomson Current Drugs, Middlesex House, 34-42 Cleveland Street, London W1T 4LB, United Kingdom.
E-mail: rebecca.erlich@current-drugs.com
SO IDrugs, (01 APR 2003), 6/4 (331-333)
CODEN: IDRUFN ISSN: 1369-7056
DT Journal; Conference Article
CY United Kingdom
LA English
SL English
AB Over the last five years, the explosion in knowledge at the molecular level in areas such as signal transduction and the cell cycle has revealed a plethora of molecular targets potentially involved in the pathogenesis of cancer. Cancer drug development is now reflecting the emerging molecular archaeology of the disease and lessons are already being learned from targeted therapies, such as gefitinib (Iressa; AstraZeneca plc), that have not achieved the expected benefits when used in combination with conventional chemotherapy in late-stage clinical trials. The disappointment with Iressa, a therapy that inhibits EGFR, has highlighted the need to rethink clinical trial designs and target populations that will benefit from such treatments. Clinical success with Celgene Corp's thalidomide (Thalomid) has demonstrated that lack of response in animals cannot reliably be extrapolated to predict the efficacy of a drug in patients. However, in the current arena of cancer therapy, it is highly unusual for a drug to reach the market where clinical responses have not been observed in phase I trials, even though this endpoint is not strictly a defining characteristic of the classic phase I trial. To achieve the full benefit from clinical trials with targeted therapies, it will be necessary to demonstrate the activity of the drug through the definition of biomarkers that are present in blood or urine and are therefore easily accessible for assay. A theme echoed throughout the 2 days of the meeting was the necessity to validate biomarkers at the preclinical stage, followed by the need to develop assays that work in the patient. Karol Sikora (AstraZeneca plc, UK), who chaired day one of the meeting proceedings, pointed out that new style phase I trials must find the dose of a drug that inhibits its target, and that the development of the technology to do so will require closer collaborations between academia, biotechnology and 'big pharma'.

L24 ANSWER 3 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.



on STN
AN 2003:36886520 BIOTECHNO
TI Immune responses to murine monoclonal antibody-B43.13 correlate with prolonged survival of women with recurrent ovarian cancer

AU Mobus V.J.; Baum R.P.; Bolle M.; Kreienberg R.; Noujaim A.A.; Schultes B.C.; Nicodemus C.F.
CS Dr. V.J. Mobus, Dept. of Obstetrics and Gynecology, Stadtische Kliniken, Gotenstrasse 6-8, 65929 Frankfurt, Germany.
E-mail: MoebusVolk@aol.com
SO American Journal of Obstetrics and Gynecology, (01 JUL 2003), 189/1 (28-36), 23 reference(s)
CODEN: AJOGAH ISSN: 0002-9378
DT Journal; Article
CY United States
LA English
SL English
AB OBJECTIVE: We evaluated the therapeutic efficiency of the murine monoclonal antibody-B43.13 in the treatment of patients with recurrent ovarian cancer. STUDY DESIGN: This was a retrospective study of immune responses and survival in 44 patients who were treated with technetium 99m-labeled monoclonal antibody-B43.13, a murine monoclonal antibody that is directed against the tumor-associated antigen CA125. Most patients were pretreated heavily. Biologic activity was quantified by the assay of immune responses to the human anti-murine antibodies against the monoclonal antibody-B43.13 variable region (Ab_2) and antibodies that target the CA 125 antigen itself (anti-CA 125 antibody). RESULTS: More than one half of patients (56.8%) survived for >12 months after the first dose of monoclonal antibody B43.13; 34.1% of the patients survived >24 months. To date, 6 of the 44 patients are alive, with survival times of 4 to 7.5 years after the start of the antibody treatment. More than 60% of the evaluable patients met predefined criteria for robust, treatment-emergent human anti-murine antibodies and Ab_2 responses, and these responses were associated with improved survival rates. Median survival time increased approximately 3-fold for human anti-murine antibody responders (22.6 months) versus nonresponders (7.2 months; $P < .0016$, log-rank test) and 2-fold for Ab_2 responders (18.3 months) versus nonresponders (9.3 months). No serious drug-associated adverse events were reported. CONCLUSION: The associations between multiple types of immune response and improved clinical outcomes suggest that monoclonal antibody-B43.13 should be further evaluated for potential use as an immunotherapy for CA125-expressing malignancies.

L24 ANSWER 4 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

Full Text | Citing References

on STN
AN 2002:35448064 BIOTECHNO
TI Monoclonal antibodies as therapeutics in oncology
AU Trikha M.; Yan L.; Nakada M.T.
CS M. Trikha, Centocor Oncology Research, 200 Great Valley Parkway, Malvern, PA 19355, United States.
E-mail: mnakada@cntus.jnj.com
SO Current Opinion in Biotechnology, (01 DEC 2002), 13/6 (609-614), 29 reference(s)
CODEN: CUOBE3 ISSN: 0958-1669
DT Journal; General Review
CY United Kingdom
LA English
SL English
AB The specificity of antibodies has been harnessed to target cancer cells and the first therapeutic antibodies for use in oncology are now finding application in the clinic. Studies are currently under way to develop new and improved antibodies. Recent developments have been made in the identification of novel targets, including the use of genomic and proteomic technologies. Several methods are also being developed to enhance antibody efficacy.

L24 ANSWER 5 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

Full Text | Citing References

on STN
AN 2002:36470165 BIOTECHNO
TI Nature's best weapons to fight cancer. Revival of human monoclonal IgM antibodies
AU Vollmers H.P.; Brandlein S.
CS Prof. H.P. Vollmers, Institut fur Pathologie, Universitat Wurzburg, Josef-Schneider-Str. 2, D-97080 Wurzburg, Germany.
E-mail: path027@mail.uni-wuerzburg.de
SO Human Antibodies, (2002), 11/4 (131-142), 123 reference(s)
CODEN: HUANFP ISSN: 1093-2607
DT Journal; Article
CY Netherlands
LA English
SL English
AB The unique features of monoclonal antibodies (specificity, effectiveness, purity and unlimited reproducibility) make them ideal tools for the specific treatment of all kind of diseases. The third generation of monoclonal antibodies for the treatment of human diseases will be, after murine and "humanised' murine immunoglobulins, fully human antibodies. The best source of human monoclonal antibodies are the antibody pools of cancer patients themselves with the best technique for generating them being conventional human hybridoma technology. This technique, will generate human monoclonal antibodies which will not only define important new targets on cancerous tissue, but will also provide the necessary therapeutic human antibodies in the fight against cancer.

L24 ANSWER 6 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

Full Text | Citing References

on STN
AN 2002:36686878 BIOTECHNO
TI Immunomodulation with antibodies: Clinical application in ovarian cancer and other malignancies
AU Nicodemus C.F.; Schultes B.C.; Hamilton B.L.
CS Dr. C.F. Nicodemus, AltaRex Corp., 610 Lincoln Street, Waltham, MA 02451, United States.
E-mail: cnicodemus@altarex.com
SO Expert Review of Vaccines, (2002), 1/1 (35-48), 95 reference(s)
CODEN: ERVXAX ISSN: 1476-0584
DT Journal; General Review
CY United Kingdom
LA English
SL English
AB This review idenfifies the role of antibodies in the field of therapeutic cancer vaccines. Ovarian cancer is used as a model to review advances in therapeutic vaccine development with a focus on antibodies as immunomodulators and antigen mimefics, highlighting research on B43.13 and ACA 125. The interaction of biological immunomodulation and chemotherapy is discussed. Requirements of antigen processing and recent advances in the field of dendritic cell biology are critical to current understanding of potent immune response induction. Future directions including use of growth factors, adjuvants and cellular therapies to enhance effects may potentiate observed effects and provide clues for application in many cancers and beyond oncology.

L24 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 2001:370629 CAPLUS
DN 136:68257
TI OvaRex (**altarex**)
AU Brukner, Ivan
CS SignalGene Inc, Montreal, QC, H2M 2N9, Can.
SO IDrugs (2001), 4(4), 457-462
CODEN: IDRUFN; ISSN: 1369-7056
PB Current Drugs Ltd.

DT Journal; General Review
LA English
AB A review. **AltaRex** is developing OvaRex (B43.13), a monoclonal antibody vaccine, for the potential treatment of ovarian cancer. Addnl., immunotherapy may be possible in other cancers expressing the CA125 antigen, such as breast and lung cancers. **AltaRex** plans to file a BLA for OvaRex with the FDA in late 2001 and for Canadian and European regulatory submissions thereafter, with possible commercialization in 2002 [365081,377828,398528]. In Dec. 2000, **AltaRex** engaged US Oncol. to participate in the company's phase II trial for the "watchful waiting" stage of ovarian cancer. The US Oncol. relationship will bring over 20 satellite sites to the OvaRex study, in addn. to 13 sites that had already begun enrolling patients [394604,384676,365081]. In 1997, **AltaRex** commenced a multicenter, placebo-controlled, double-blind, randomized phase IIb trial in the US for advanced ovarian cancer [230067,331744,344248]. In the following year, **AltaRex** submitted the IND to the FDA for this trial [303667]. Complete anal. of the data is expected by the first half of 2001 [291312,310071,344248]. If an NDA is filed before the end of 2001, approval by mid-2002 is possible [344248]. OvaRex has been awarded Fast Track designation for advanced ovarian cancer [310071] and Orphan Drug status in the US [230067].
RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2

Full Text | Citing References

AN 1999:222222 CAPLUS
DN 131:72456
TI Immunotherapy of human ovarian carcinoma with OvaRex MAb-B43.13 in a human-PBL-SCID/BG mouse model
AU Schultes, Birgit C.; Zhang, Chengsheng; Xue, Lanny Y.; Noujaim, Antoine A.; Madiyalakan, Ragupathi
CS AltaRex Corp., Edmonton, AB, T6G 2N8, Can.
SO Hybridoma (1999), 18(1), 47-55
CODEN: HYBRDY; ISSN: 0272-457X
PB Mary Ann Liebert, Inc.
DT Journal
LA English
AB The monoclonal antibody (MAb) B43.13, binding to the ovarian cancer-assocd. antigen CA125, has been injected into >200 patients with ovarian cancer to detect recurrence of the disease. The follow-up of the patients revealed surprisingly long survival spans for several patients despite high CA125 levels. To investigate the therapeutic effectiveness of OvaRex MAb-B43.13 (**AltaRex**, Edmonton, Canada) under well-controlled conditions, the antibody was tested in a human-PBL-SCID/BG mouse model with CA125 pos. human ovarian cancer cells. Mice were reconstituted with human peripheral blood lymphocytes (PBL, normal donors) by i.p. (IP) injection of 2-3×107 PBL/mouse. OvaRex MAb-B43.13 was administered at 100 μg/mouse in phosphate buffered saline (PBS), in 3 different exptl. set-ups. An isotype-matched control antibody (MOPC21 or MAb-170) and PBS injection served as controls. The ovarian cancer cell line NIH:OVCAR-NU-3 was injected IP at 1×106 cells/mouse or s.c. (SC) at 4×106 cells/mouse. Human-PBL-SCID/BG mice were either immunized before injection of tumor cells, along with tumor cells or after small tumors were established (2 wk after transplantation). Antibody injections were repeated twice in 2-wk intervals. Functional and cellular characterization of serum and PBL from these mice demonstrated the successful engraftment of a human immune system in those mice. All 3 expts. showed that OvaRex MAb-B43.13 treatment could (1) delay or prevent development of tumors; (2) reduce the size of small established tumors (SC tumor injection) or suppress ascites formation; (3) delay tumor growth when injected prior to tumor implantation; and (4) prolong the survival of the mice (IP tumor injection).
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 9 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

Full Text | Citing References

on STN

AN 1998:28384465 BIOTECHNO

TI Summary report on the ISOBM TD-6 workshop: Analysis of 20 monoclonal antibodies against Sialyl Lewis(a) and related antigens. Montreux, Switzerland, September 19-24, 1997

AU Rye P.D.; Bovin N.V.; Vlasova E.V.; Molodyk A.A.; Baryshnikov A.; Kreutz F.T.; Garinther W.I.; Schultes B.C.; Noujaim A.A.; Madiyalakan R.; Magnani J.; Nilsson O.; Nilsson K.; Nustad K.; Norum L.; Bells H.; Caoh Y.; Suresh M.R.; Very D.L.; Freeman J.V.; Yeung K.K.; Hilgers J.

CS Dr. N.V. Bovin, Shemyakin Inst. Bioorganic Chemistry, Russian Academy of Sciences, Ul. Miklukho-Maklaya, 16/10, 117871 GSP-7 Moscow V437, Russian Federation.

SO Tumor Biology, (1998), 19/5 (390-420), 13 reference(s)
CODEN: TUMBEA ISSN: 1010-4283

DT Journal; Article

CY Switzerland

LA English

SL English

AB The ISOBM TD-6 Workshop is the first international workshop on monoclonal antibodies against the Sialyl Lewis(a) (SLe(a)) antigen. Eight research groups participated in a blind study to characterize the epitope binding, relative affinity and performance in immunoradiometric assays, of a panel of 20 monoclonal antibodies. The antibodies were tested against a diverse panel of neoglycoconjugates, purified antigens and human serum pools from gastrointestinal malignancies. Epitope specificities were determined for the majority of antibodies in the panel. Cross-reactivity with related saccharide structures was noted in several antibodies. Overall, the results of the TD-6 Workshop show further development of SLe(a) immunoassays may yield yet more specific assays for the detection and management of gastrointestinal and other malignancies.

L24 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 1999:506028 CAPLUS

DN 132:90130

TI The effects of circulating antigen on the pharmacokinetics and radioimmunoscintigraphic properties of 99mTc labelled monoclonal antibodies in cancer patients

AU McQuarrie, S. A.; Riauka, T.; Baum, R. P.; Sykes, T. R.; Noujaim, A. A.; Boniface, G.; MacLean, G. D.; McEwan, A. J. B.

CS Fac. Pharmacy and Pharmaceutical Sci., Univ. Alberta, Edmonton, Can.

SO Journal of Pharmacy & Pharmaceutical Sciences [Electronic Publication] (1998), 1(3), 115-125
CODEN: JPPSFY; ISSN: 1482-1826
URL: http://www.ualberta.ca/~csps/JPPS1(3)/S.McQuarrie/Mabs-McQuarrie.pdf

PB Canadian Society for Pharmaceutical Sciences

DT Journal; (online computer file)

LA English

AB This article reports the pharmacokinetics, radiation dosimetry and radioimmunoscintigraphy (RIS) of two 99mTc-labeled monoclonal antibodies (MAb) used to detect cancer. The effects of circulating antigen in female cancer patients are explored and their effects on the ability of these MAbs to effectively perform as RIS agents noted. To illustrate the effects of circulating antigen, data using MAb B43.13 (OVAREX, **AltaRex** Corp., Waltham, MA, USA) from a Pilot study in ovarian cancer patients are presented. The results from a Phase II study of MAb 170H.82 (Tru-Scint AD, BIOMIRA INC., Edmonton, Alberta, Canada) in patients with primary and locally recurrent breast cancer were used to portray the biodistribution patterns when no circulating antigen is present. Data from planar gamma camera images were obtained for both groups and used for pharmacokinetic and radiation dosimetry analyses. A pharmacokinetic anal. indicated a

shorter residence time and higher clearance of 99mTc-MAb-B43.13 that was ascribed in part to the circulating CA 125 antigen in this group of ovarian cancer patients. These clearance patterns resulted in acceptable, though higher radiation doses to the spleen and urinary bladder wall for these patients when compared to the MAb-170H.82 group. Both MAbs were found to produce acceptable radioimmunoscintigraphic images.

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 11 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

Full Text | Citing References

on STN
AN 1997:27360022 BIOTECHNO
TI Radiolabeling of monoclonal antibody B43.13 with Rhenium-188 for immunoradiotherapy
AU Sykes T.R.; Somayaji V.V.; Bier S.; Woo T.K.; Kwok C.S.; Snieckus V.; Noujaim A.A.
CS T.R. Sykes, Pharmacy/Pharmaceutical Sci. Faculty, University of Alberta, Edmonton, Alta., Canada.
SO Applied Radiation and Isotopes, (1997), 48/7 (899-906), 35 reference(s)
CODEN: ARISEF ISSN: 0969-8043
PUI S0969804397000250
DT Journal; Article
CY United Kingdom
LA English
SL English
AB In this study we report a novel method for direct radiolabeling of monoclonal antibody B43.13 (MAb-B43.13) with ^{188}Re and have evaluated the product's radiochemical of biochemical, immunochemical and selected biological properties. ^{188}Re - MAb - B43.13 was readily prepared by the addition of generator produced perrhenate to a preformulated antibody vial after an optima amount of supplemental stannous ion, in the form of stannous tartrate, was added. The final radiolabeled, product retained its biochemical purity (as determined by size-exclusion HPLC and R/NR-SDS-PAGE), its immunoreactivity (as determined by immunoassay) and presented with a typical stability (in the presence of serum and cysteine) and biodistribution (in tumored mice) profile. The evaluation of the product for immunoradiotherapy of ovarian cancer in a clinical setting requires further studies.

L24 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text | Citing References

AN 1997:177231 CAPLUS
DN 126:213678
TI Porous exchanger and water vapor pump: The **ALTAREX** boiler
AU Guillet, R.; Brunel, G.; Grehier, A.; Viltard, J. C.
CS Gaz de France, Fr.
SO Proceedings of the International Gas Research Conference (1996), Volume Date 1995, (Vol. 2), 2783-2792
CODEN: PGRCDV; ISSN: 0736-5721
PB Government Institutes
DT Journal
LA English
AB A porous membrane exchanger in the **ALTAREX** boiler constitutes a new technol. approach to water vapor pumps. This mass and heat exchanger uses the combustion air as a secondary heat sink for recycling the residual latent and sensible energy which is usually lost up the chimney in combustion-type heaters. This means that the **ALTAREX** boiler can recover all of the higher calorific value of natural gas. In addn. to an increased energy efficiency, the boiler offers the advantages of combustion with high water-vapor enriched air, which generates very little NOx formation. This energy efficiency and low pollution make the **ALTAREX** boiler and other spin-off technol. systems (i.e., dryers, direct contact heaters, gas turbines, etc.) world-wide leaders in heat energy

prodn. from fuel combustion, esp. natural gas.

```
=> s Noujaim, Antoine
L25           0 NOUJAIM, ANTOINE

=> s baum, richard
L26           0 BAUM, RICHARD

=> s (antoine noujaim)
L27           0 (ANTOINE NOUJAIM)

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